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Catheter Ablation of Atypical Atrial Flutter and Atrial Tachycardia Within the Coronary Sinus After Left Atrial Ablation for Atrial Fibrillation

Aman Chugh, MD, Hakan Oral, MD, FACC, Eric Good, DO, Jihn Han, MD, Kamala Tamirisa, MD, Kristina Lemola, MD, Darryl Elmouchi, MD, David Tschopp, MD, Scott Reich, MD, Petar Igic, MD, Frank Bogun, MD, Frank Pelosi, Jr, MD, FACC, Fred Morady, MD, FACC

Ann Arbor, Michigan

OBJECTIVES The goal of this study was to describe the prevalence and ablation of coronary sinus (CS) arrhythmias after left atrial ablation for atrial fibrillation (AF).

BACKGROUND The CS has been implicated in a variety of supraventricular arrhythmias.

METHODS Thirty-eight patients underwent mapping and ablation of atypical flutter that developed during (n = 5) or after (n = 33) ablation for AF. Also included were two patients with focal CS arrhythmias that occurred during an AF ablation procedure. A tachycardia was considered to be originating from the CS if the post-pacing interval in the CS matched the tachycardia cycle length and/or if it terminated during ablation in the CS.

RESULTS Among the 33 patients who developed atypical flutter late after AF ablation, 9 (27%) were found to have a CS origin. Overall, 16 of the 40 patients in this study had a CS arrhythmia. The tachycardia was macro–re-entrant in 14 patients (88%) and focal in two patients. Radiofrequency ablation with an 8-mm-tip catheter was successful in 15 patients (94%) without complication. In eight patients (50%), ≥45 W was required for successful ablation. Thirteen of the 15 patients (87%) with a successful ablation acutely remained arrhythmia-free during 5 ± 5 months of follow-up.

CONCLUSIONS The musculature of the CS serves as a critical component of the re-entry circuit in approximately 25% of patients with atypical flutter after ablation for AF. The CS may also generate focal atrial arrhythmias that may play a role in triggering and/or maintaining AF. Catheter ablation of these arrhythmias in the CS can be performed safely. (J Am Coll Cardiol 2005;46:83–91) © 2005 by the American College of Cardiology Foundation

The musculature of the coronary sinus (CS) has been implicated in a variety of arrhythmias, including those mediated by accessory pathways (1), and focal (2,3) and macro–re-entrant (4) atrial tachycardias. Electrical disconnection of the CS from the left atrium has also been shown to decrease the probability of induction of atrial fibrillation (AF) in patients undergoing pulmonary vein isolation (5). However, the role of the CS in arrhythmias after left atrial ablation has not been described. The purpose of this study was to determine the prevalence and mechanisms of atrial arrhythmias originating from the CS in patients who underwent left atrial ablation for AF.

METHODS

Patient characteristics. The study group (Fig. 1) consisted of 38 patients who underwent mapping and ablation of atypical flutter that developed either during (n = 5) or late after (n = 33) left atrial ablation for AF. In the latter group of 33 consecutive patients, the atypical flutter was diagnosed a mean of 8 ± 10 weeks after ablation of AF, and the flutter ablation procedure was performed a mean of 8 ± 4 months after the ablation procedure for AF. Also included in this study were two patients with a focal atrial arrhythmia originating in the CS that occurred during an ablation procedure for AF.

The musculature of the CS was considered to be a component of the re-entry circuit if the post-pacing interval (PPI) in the CS was within 20 ms of the tachycardia cycle length or if the arrhythmia terminated during energy delivery in the CS. Table 1 describes the clinical characteristics of the 40 patients.

Left atrial ablation for AF. Antiarrhythmic medications were discontinued at least five half-lives before the procedure, with the exception of amiodarone. In patients with paroxysmal AF, amiodarone was discontinued ≥6 weeks before the procedure, and in patients with persistent or chronic AF, amiodarone therapy was not discontinued before the procedure. The ablation procedure was performed in the fasting state after written informed consent was obtained. Catheters were inserted into the right femoral vein and/or the right internal jugular vein. A quadripolar electrode catheter was positioned within the CS for atrial pacing. After transeptal catheterization, heparin was infused to maintain an activated clotting time of 300 to 350 s. A deflectable quadripolar catheter (EP Technologies, Inc., Sunnyvale, California) was placed in the coronary sinus for atrial pacing (EP-3 Clinical Stimulator, EP MED Systems, Inc., West Berlin, New Jersey).

Left atrial circumferential ablation was performed as

From the Division of Cardiology, University of Michigan Hospitals, Ann Arbor, Michigan.

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Abbreviations and Acronyms
AF = atrial fibrillation
CS = coronary sinus
ECG = electrocardiogram
PPI = post-pacing interval

previously described (6). An 8-mm-tip deflectable ablation catheter (Navistar, Biosense Webster, Diamond Bar, California) was used to create a three-dimensional replica of the left atrium with an electroanatomic mapping system (CARTO, Biosense Webster). Tubular models of the pulmonary veins and the outline of the mitral valve annulus were also depicted. Radiofrequency current was applied with a target temperature of 50°C to 55°C and a power of 50 to 70 W (Stockert 70 RF generator, Biosense Webster). Left atrial ablation was performed 1 to 2 cm from the pulmonary vein ostia to encircle the left- and right-sided pulmonary veins. The encircling ablation lines were then connected with one to two ablation lines in the posterior left atrium. An ablation line also was created between the inferior aspect of the left-sided encircling ablation line and the mitral annulus. After the recognition of the possibility of left atrial-esophageal fistula (7), the posterior line was moved to the anterior portion of the roof and lower power and/or temperature settings were used on the posterior wall. The end point of ablation was voltage abatement of the local atrial electrogram by >80% or to <0.1 mV.

If the procedure was performed during AF and the patient converted to sinus rhythm during ablation, atrial pacing was performed on five occasions for 10 s at cycle lengths of 200 to 220 ms and programmed atrial stimulation was performed with a single atrial extrastimulus to determine whether AF or another arrhythmia was inducible. If after ablation the patient remained in AF, ibutilide was administered or transthoracic cardioversion was performed. Atrial flutter was defined as a macro–re-entrant, regular, atrial tachycardia with a cycle length of ≥200 ms and a consistent atrial activation sequence. It was considered atypical if the PPI at the cavotricuspid isthmus exceeded the cycle length of the tachycardia by >30 ms. Macro–re-entry was confirmed by observing that atrial electrograms spanned the cycle length of the tachycardia. An atrial tachycardia was considered focal if there was a centrifugal pattern of spread from a focal source.

Table 1. Clinical Characteristics of the Study Patients

Number of patients	40
Age (yrs)	55 ± 9
Males/females	26/14
Duration of AF (yrs)	6 ± 7
Paroxysmal/persistent AF	22/18
LV ejection fraction	0.52 ± 0.12
Left atrial diameter (mm)	43 ± 6
Structural heart disease	8

Data are shown as mean ± SD.
AF = atrial fibrillation; LV = left ventricular.

Ablation was performed in the cavotricuspid isthmus in patients with a history of typical atrial flutter, and in patients in whom cavotricuspid isthmus-dependent atrial flutter was induced by atrial pacing (8).

Mapping of CS arrhythmias. An activation map of the atypical flutter was performed with an electroanatomic mapping system. A CS origin was suspected if the entire cycle length of the macro–re-entrant tachycardia could not be accounted for by a left atrial activation map or if the CS atrial electrogram occurred in mid-diastole. Entrainment mapping was performed to identify sites within the re-entry circuit (9). During pacing from the CS, the pacing output was gradually decreased to avoid concomitant capture of the adjacent left atrium. Sites harboring split, fragmented, or diastolic potentials were labeled on the activation map. Focal arrhythmias were mapped by identifying the site of earliest atrial activation with respect to the P-wave on the 12-lead electrocardiogram (ECG) and/or with an electroanatomic mapping system. Each pulmonary vein was analyzed during tachycardia with a mapping catheter to rule out

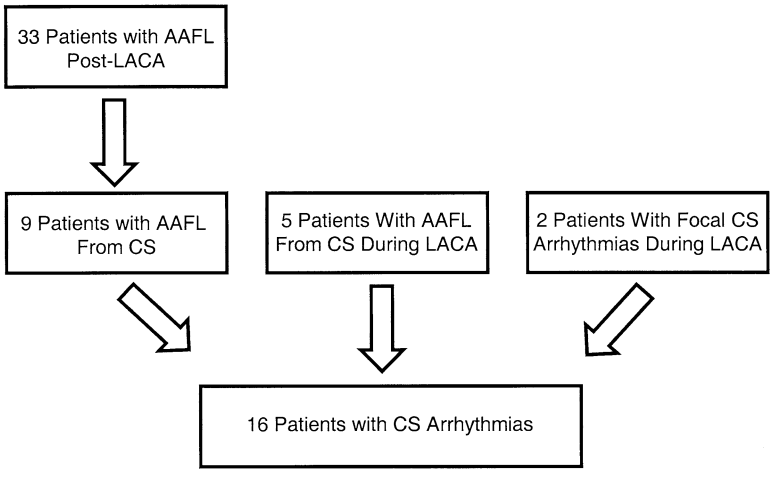

Figure 1. A flow chart showing how the patients were selected for the study. AAFL = atypical atrial flutter; CS = coronary sinus; LACA = left atrial circumferential ablation.

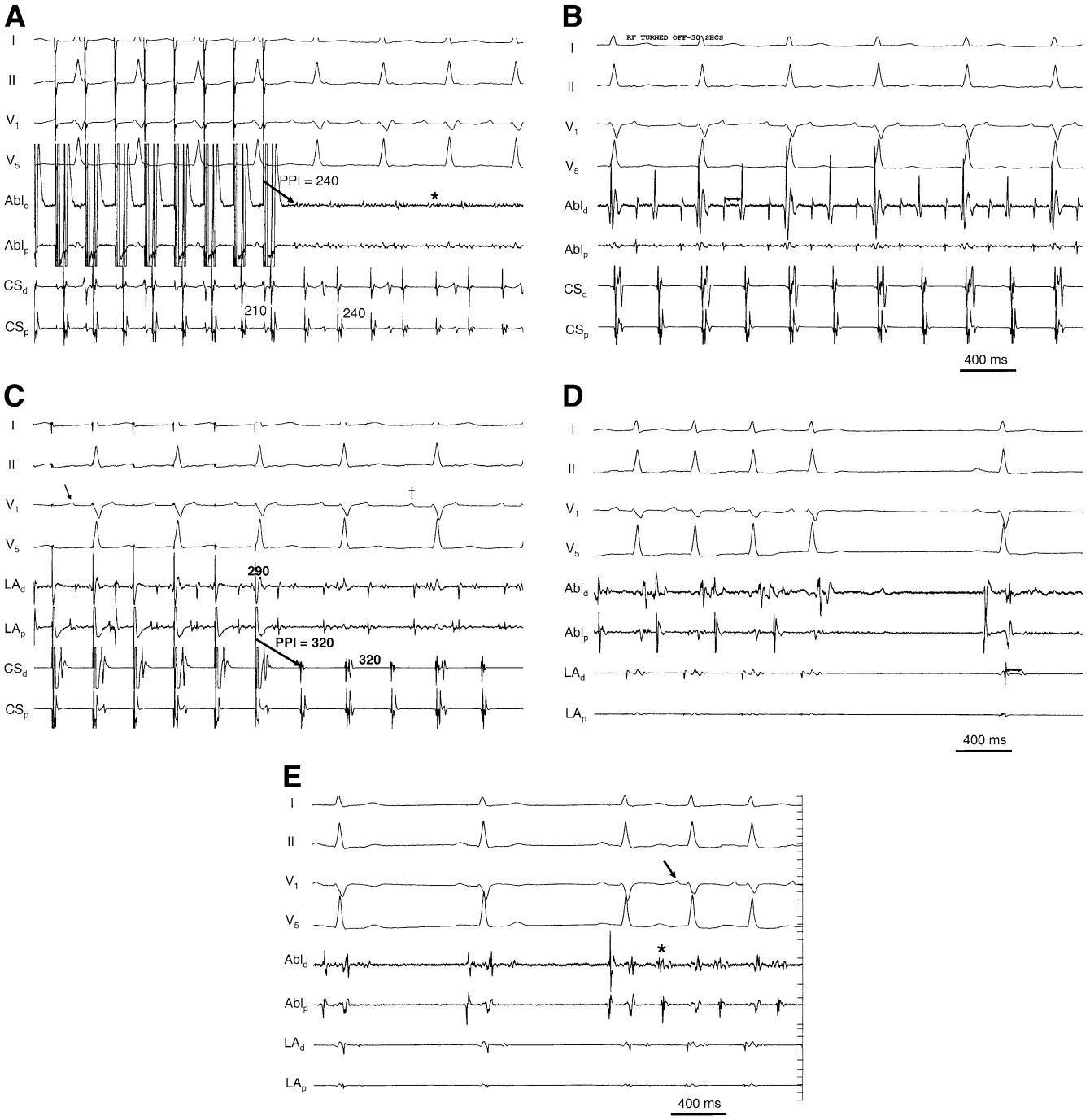

Figure 2. **(A)** Entrainment mapping from the mitral isthmus in Patient #13, whose electrocardiogram (ECG) is shown in Figure 4C. The tachycardia is accelerated to the pacing rate of 210 ms and the post-pacing interval (PPI) matches the tachycardia cycle length (TCL), confirming that the tachycardia is mitral isthmus-dependent. Note the fragmented potential **(*)** with diastolic activity recorded by the distal pole of the ablation catheter (Abl_d), consistent with a gap in the mitral isthmus line. Also shown are ECG leads I, II, V_1, and V_5, and bipolar electrograms recorded by the distal and proximal poles of the ablation catheter (Abl_d and Abl_p) and a catheter placed in the coronary sinus (CS_d and CS_p). **(B)** The result of radiofrequency (RF) current application at the mitral isthmus in the same patient as in Figure 2A. After 30 s of current application at the site where the PPI matched the TCL, widely split double potentials **(arrow)** are now recorded. The tachycardia also slows from 240 ms to 320 ms without a change in the P-wave morphology. The electrogram recorded by the CS catheter is now in mid-diastole. **(C)** Another example of concealed entrainment during pacing from the distal CS in the same patient as in Figures 2A and 2B. The tachycardia is accelerated to the pacing rate, and PPI matches the TCL. The P-wave morphology during pacing **(small arrow)** is identical to that during tachycardia **(†)**. Also note the double potentials as recorded by the left atrial catheter (LA_d), which was positioned at the mitral isthmus. **(D)** The effect of RF current application in the CS in the same patient as above. The tachycardia had slowed from 320 ms to 400 ms before termination. Also, note the double potential **(arrow)** recorded by the left atrial catheter (LA_d) at the mitral isthmus even during sinus rhythm. The first component of the double potential occurs simultaneously with the QRS suggestive of marked delay in lateral left atrial activation. **(E)** An example of immediate recurrence of atrial flutter in the same patient as above. The same tachycardia reappeared repeatedly within seconds of discontinuation of RF energy with an identical cycle length. Further energy application in the distal CS at a higher power terminated the tachycardia without recurrence or inducibility. Note that the P-wave morphology of the premature atrial depolarization that triggers the tachycardia **(arrow)** is very similar to that during tachycardia, suggesting that the sites responsible for initiating and maintaining the tachycardia are located in the CS.

the possibility of a pulmonary vein tachycardia with exit block.

Radiofrequency ablation in the CS. Before ablation in the CS, radiofrequency energy was delivered first in the left atrium at sites where the PPI was closest to the tachycardia cycle length. For focal tachycardias, target sites were selected on the basis of earliest endocardial atrial activation. Catheter ablation in the CS was performed with an 8-mm-tip ablation catheter (Navistar, Biosense Webster). If a large ventricular electrogram was recorded with the ablation catheter, high-output pacing was performed to rule out ventricular capture before ablation. The initial power and temperature settings were 35 W and 50°C. If the tachycardia did not terminate, power and temperature were gradually increased to a maximum of 70 W and 55°C, as long as the impedance did not exceed 110 ohms and the delivered power remained near the maximum power setting. The catheter was slowly withdrawn every 10 to 15 s. Ablation was not performed at sites where the impedance was >110 ohms. To avoid overheating and "tissue pops," energy applications were discontinued whenever the delivered power was <10 W. Energy applications also were discontinued at sites where ablation was painful. Coronary angiography was not performed in any of the study patients.

Procedural success was defined as termination of the tachycardia by radiofrequency application and the inability to reinduce the tachycardia by rapid atrial pacing and programmed atrial stimulation. Pacing was repeated during infusion of 4 μg/min of isoproterenol.

Postablation management. After the procedure, all patients received anticoagulation with intravenous heparin overnight and were discharged the next day. The patients were treated with low-molecular-weight heparin for five days and with warfarin for at least three months. Patients with paroxysmal AF were treated for three months with a beta-blocker, calcium-channel blocker, and/or the same antiarrhythmic medication they had been taking before the procedure. Patients with chronic AF were treated with amiodarone for three months after the procedure. Patients who underwent a repeat procedure for atypical flutter were not prescribed antiarrhythmic medications on discharge.

Follow-up. Patients were seen in an outpatient clinic at three and at six to nine months after the procedure. If the patient remained free of symptomatic AF, antiarrhythmic and rate-control medications were discontinued at the three-month appointment. All patients were instructed to contact a nurse if symptoms suggestive of AF recurred, and these patients underwent an ECG and/or 30 days of monitoring with a continuous loop recorder.

If atypical flutter developed during follow up, the initial management consisted of rate control and anticoagulation. Antiarrhythmic medications were discontinued to rule out the possibility of proarrhythmia (10). If the arrhythmia persisted beyond three to six months, another procedure was recommended.

Statistical analysis. Continuous variables are expressed as mean ± 1 SD.

RESULTS

Among the 33 patients who underwent mapping and ablation of atypical flutter that occurred late after ablation of AF, the CS was a critical component of the re-entry circuit in 9 patients (27%) (Fig. 1). In four of these nine patients (44%), entrainment mapping indicated that the mitral isthmus also was part of the re-entry circuit (Fig. 2), but endocardial ablation was unsuccessful and termination of the flutter required ablation in the CS. The successful site within the CS in these four patients was 1 to 2 cm from the mitral isthmus ablation line. These 4 patients comprised 19% of 21 patients who were found to have a mitral isthmus-dependent flutter.

An additional five patients developed a macro–re-entrant tachycardia involving the CS during an AF ablation procedure. Based on entrainment mapping, the mitral isthmus was not part of the re-entry circuit in these five patients.

Another two patients had a focal atrial arrhythmia originating in the CS that occurred during left atrial ablation for AF. Therefore, a total of 16 patients were found to have arrhythmias involving the musculature of the CS after left atrial ablation for AF.

Description of CS arrhythmias. The mechanism of the CS arrhythmias was macro–re-entry in 14 of the 16 patients (88%) (Table 2). The mean cycle length of the macro–re-entrant flutters was 262 ± 28 ms (range 220 to 310 ms). A perfect PPI was found in the proximal CS in four patients (28%), mid-CS in five patients (36%), and distal CS in five patients (36%) (Fig. 3).

Two patients were found to have a focal ectopic rhythm or tachycardia (cycle length 745 ms and 315 ms, respectively) originating within the CS during the index procedure for AF. Based on activation mapping, these focal arrhythmias arose in the distal or mid-coronary sinus.

ECG characteristics of coronary sinus arrhythmias. Table 3 shows the P-wave polarity on the 12-lead ECG of the arrhythmias that were ablated in the CS. None of the ECGs showed inverted P waves in the inferior leads (Fig. 4). Specifically, 12 of the 16 patients (75%) showed positive P waves in the inferior leads. Eleven of the 16 patients (69%) patients showed isoelectric P waves in lead I, and 10 of the 16 (63%) patients had negative P waves in lead aVL. All but two patients (88%) had upright P waves in lead V_1. A transition from positive to negative polarity was never observed across the precordial leads.

Results of catheter ablation. Based on entrainment or activation mapping, the target site was the proximal CS in four patients (25%), mid-CS in six patients (37%), and distal CS in six patients (38%). Catheter ablation was successful in terminating the tachycardia in 15 of the 16 patients (94%), and in no case was it reinducible. In one patient (#8), radiofrequency current was not delivered in the

Table 2. Procedural Characteristics of Patients Who Underwent Catheter Ablation of Coronary Sinus Arrhythmias

Pt. No.	Timing	Cycle Length (ms)	Mechanism	CS Location	Power
1	Follow-up	285	Macro–re-entrant	Proximal	Low
2	Acute	745	Focal	Distal	Low
3	Acute	245	Macro–re-entrant	Proximal	Low
4	Acute	310	Macro–re-entrant	Proximal	Low
5	Follow-up	260	Macro–re-entrant	Distal	Low
6	Acute	300	Macro–re-entrant	Mid	High
7	Follow-up	240	Macro–re-entrant	Proximal	High
8	Acute	250	Macro–re-entrant	Mid	Low
9	Follow-up	220	Macro–re-entrant	Distal	Low
10	Follow-up	230	Macro–re-entrant	Mid	Med
11	Follow-up	275	Macro–re-entrant	Mid	Med
12	Acute	275	Macro–re-entrant	Mid	Med
13	Follow-up	235	Macro–re-entrant	Distal	High
14	Follow-up	300	Macro–re-entrant	Distal	Low
15	Acute	315	Focal	Mid	Med
16	Follow-up	240	Macro–re-entrant	Distal	High

"Acute" refers to tachycardias that were ablated during the procedure for atrial fibrillation. "Follow-up" refers to tachycardias that developed during follow-up after the left atrial ablation procedure for atrial fibrillation and were ablated during a repeat procedure. Power during radiofrequency application in the coronary sinus: low ≤35 W; medium = 36 to 45 W; high = 46 to 70 W.

CS = coronary sinus.

CS because of a high impedance of 135 ohms. Energy delivery at the corresponding site in the left atrium was unsuccessful. In one patient (#7) in whom entrainment mapping yielded a perfect PPI in the proximal CS, radiofrequency application failed to terminate the tachycardia despite increasing the power to 70 W. Energy delivery at the corresponding site in the left atrium, where the PPI was within 20 ms of the tachycardia cycle length, also was unsuccessful. Tachycardia termination in this patient required ablation at the low right atrial septum, after which the tachycardia was no longer inducible. The cavotricuspid isthmus had been excluded as part of the circuit by entrainment mapping.

In one patient (#2) with a focal CS arrhythmia, the earliest endocardial activation was at the mitral isthmus. Ablation at this site was ineffective. The earliest activation (−100 ms relative to the onset of the P-wave) was found in the distal CS, with the tip of the ablation catheter oriented superiorly. Energy application led to acceleration and then permanent elimination of the tachycardia. Before ablation, AF had been readily inducible in both patients with a focal CS arrhythmia. After elimination of the CS arrhythmias, AF was no longer inducible in either patient.

In two patients with mitral isthmus-dependent flutter who required ablation in the CS for termination, left atrial ablation did not affect the cycle length of the tachycardia. Left atrial ablation increased the cycle length by 30 ms in one patient (#9) and by 80 ms in another patient (#13) before termination with CS ablation. In the latter case, the PPI from the distal CS was perfect before ablation at the mitral isthmus.

Radiofrequency energy delivery in the CS. In 8 of 16 patients (50%), the CS arrhythmia terminated using the initial power and temperature setting of 35 W and 50°C. In four patients (25%) ≥45 W was required and in four patients (25%) ≥55 W was required for tachycardia termination. There were no instances of abrupt impedance increase or power drop.

Complications. No patient in this series experienced perforation or clinically apparent coronary artery complications. The ECGs recorded the morning after the procedure was unchanged from baseline in all patients. Atypical chest pain developed in one patient (#5) six months after the CS ablation procedure. Extensive testing, including exercise perfusion study, echocardiogram, and cardiac chest tomography (to evaluate for pulmonary vein or coronary artery/CS stenosis, esophageal injury, and pulmonary embolism) was unrevealing.

Follow-up. Among the 15 patients who underwent successful ablation of the CS arrhythmia, 13 patients (87%) have remained asymptomatic without antiarrhythmic medications over a course of 5 ± 5 months. Recurrent AF developed in one patient, and a different tachycardia developed in one patient.

DISCUSSION

Main findings. Atrial flutter that arises after left atrial circumferential ablation of AF uses the musculature of the coronary sinus in approximately 25% of cases. Although the mitral isthmus was the most common target in patients with atypical flutter after left atrial ablation, tachycardia termination required radiofrequency application in the CS in approximately 20% of patients with mitral isthmus-dependent flutter. Macro–re-entry was the most common mechanism of tachycardias arising in the CS, and a small percentage were focal in nature. Although low power is recommended when ablating in the CS (11), relatively high power and temperature settings were required in one-half of the patients. Radiofrequency ablation within the CS was highly effective and did not cause any complications.

These findings show that the musculature of the CS may

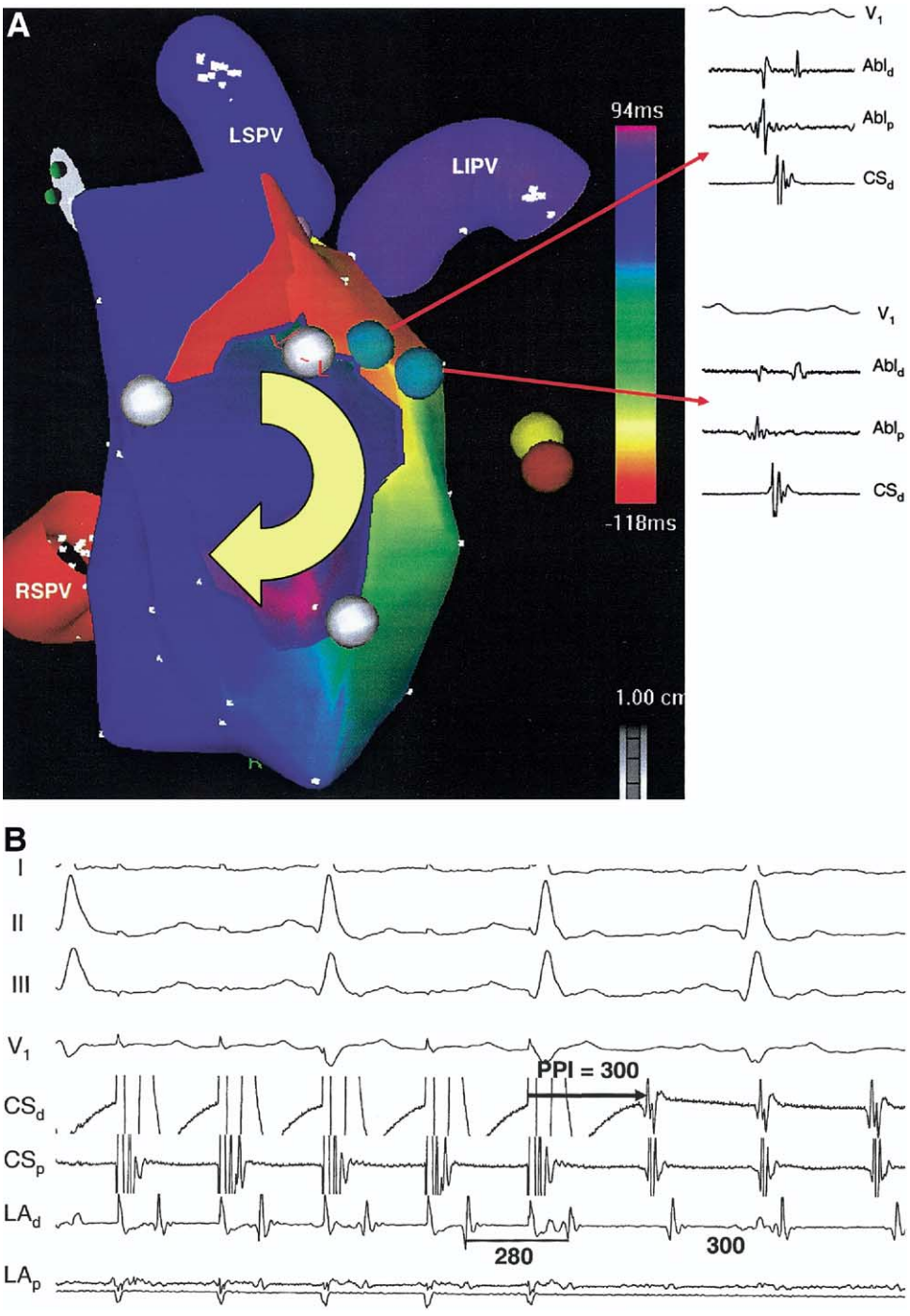

Figure 3. (A) A left atrial activation map from Patient #14 shown in a left anterior oblique projection with a caudal tilt. Although the activation map suggests clockwise mitral isthmus-dependent flutter, a corridor of widely split double potentials **(blue tags)** were noted along the isthmus indicative of local conduction block **(red arrows)**. The cycle length of the tachycardia was 300 ms, and only 212 ms could be accounted for by the activation map. Because of local block, radiofrequency (RF) application was not delivered in the left atrium. The **yellow tag** represents the site in the CS where the PPI matched TCL (Fig. 3B). The site of successful ablation within the CS is depicted by the **red tag**. **(B)** Entrainment mapping from the distal CS in the same patient as in Figure 3A. The tachycardia is accelerated to the pacing rate of 280 ms. The PPI exactly matches the TCL, indicating that the distal CS is part of the re-entrant circuit. A single application of RF current terminated the tachycardia, which was no longer inducible. Abbreviations as in Figure 2.

play an important role in generating supraventricular arrhythmias in a significant proportion of patients in whom atypical flutter develops after left atrial ablation for AF.

Mechanisms of CS tachycardias. Atypical flutter that arises after linear left atrial ablation may often be caused by gaps in the ablation lines (12,13). Because catheter ablation was not performed in the CS for AF in any of the patients, it may be difficult at first glance to invoke lesion discontinuity as an explanation for macro–re-entrant CS tachycardias. It is well known that segments of the CS are in

Table 3. Electrocardiographic Characteristics of Patients With Coronary Sinus Arrhythmias

Pt. No.	I	II	III	aVR	aVL	aVF	V_1	V_2	V_3	V_4	V_5	V_6
1	=	+	+	−	−	+	+/−	+	+	+	+	+
2	Multi	Multi	=	+	=	=	+	=	=	Multi	=	Multi
3	=	+	+	−	−	+	+	+	+	+	+	+
4	=	+	+	−	−	+	+	+	+	+	+	+
5	+	+	+	−	=	+	+	+	+	+	+	+
6	=	+	+	−	−	+	+	+	+	+	+	+
7	=	+	+	+/−	−	+	+	+	+	+	+	+
8	+	+	+	−	−	+	+	+	+	+	+	+
9	−/+	=	=	=	−/+	=	−/+	−/+	−/+	−/+	−/+	−/+
10	=	+	+	−	−	+	+	+	+	+	+	+
11	=	=	=	−	=	=	+	+	+	=	=	=
12	=	+	+	−	−	+	+	+	+	+	+	+
13	=	Multi	Multi	=	=	Multi	+	+	=	=	=	=
14	+	+	+	−	−	+	+	+	+	+	+	+
15	=	+	+	=	−	+	+	+	+	+	+	+
16	=	+	+	−	=	+	+	+	+	+	+	+

+, − =, and −/+ refer to upright, inverted, isoelectric, and biphasic P waves, respectively. "Multi" refers to complex P-wave morphology with multiple components.

electrical continuity with the left atrium by virtue of discrete muscular connections (14). Furthermore, double potentials suggestive of conduction delay have been shown in the distal CS, particularly in patients with a history of AF (15). The mitral isthmus ablation line created during the index procedure for AF is anatomically in close proximity to the course of the distal CS. Radiofrequency current application at the inferoposterior left atrium may have further slowed conduction along the CS and facilitated macro–re-entry.

Although in one case entrainment mapping identified the proximal CS as being within the re-entry circuit, energy delivery at the low right atrial septum was required to terminate tachycardia. This observation implies that right atrial-CS connections may also serve as the substrate for atypical flutter after left atrial ablation.

In one patient (#13) with mitral flutter, a single application at the mitral isthmus resulted in widely split double potentials and significant slowing of the tachycardia (240 ms to 320 ms) without a change in the P-wave morphology (Fig. 2B). The fact that the PPI from the distal CS matched the tachycardia cycle length before and after endocardial ablation suggests the existence of a double-loop tachycardia

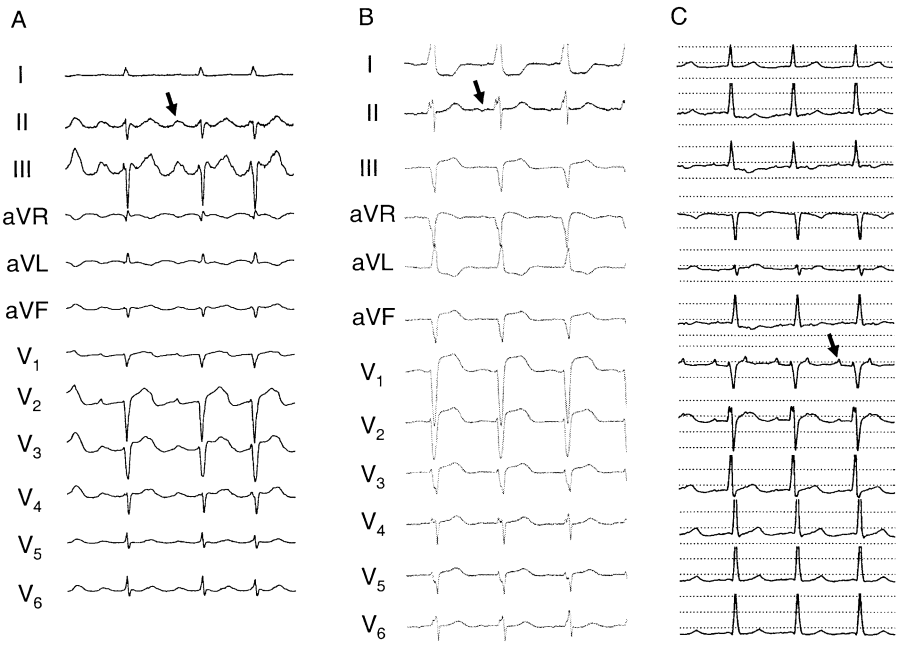

Figure 4. (A) A 12-lead electrocardiogram (ECG) from Patient #1. Note the upright P waves in the inferior leads **(arrow)** and the lack of transition across the precordial leads. The paper speed is 25 mm/s. **(B)** A 12-lead ECG from Patient #2. Note the multicomponent P waves in lead II **(arrow)**. The paper speed is 25 mm/s. **(C)** A 12-lead ECG from Patient #13. The P waves in lead V_1 are upright **(arrow)**. The paper speed is 25 mm/s. Abbreviations as in Figure 2.

in which the "faster" loop was using both the mitral isthmus and the CS and "slower" loop was primarily using the musculature of the CS.

In addition to serving as a site for maintaining macro–re-entrant tachycardias, the CS may also be a source of focal discharges. The fluoroscopic position of the ablation catheter in the distal CS may implicate the ligament of Marshall as a possible arrhythmia source in Patient #2 (16). Presumably, left atrial ablation eliminated fibrillatory conduction, unmasking the focal arrhythmia. After abolition of the focal arrhythmia, AF was no longer inducible in the two patients with focal arrhythmias, suggesting that the CS may have been serving as a driver of AF.

ECG characteristics of CS arrhythmias. Because of the inferoposterior location of the CS, one would expect the ECG during a CS arrhythmia to show inverted P waves in the inferior leads. However, P waves in the inferior leads most commonly were upright. This may be because the extensive left atrial ablation that had previously been performed resulted in altered activation of the left atrium. The majority of the patients also showed positive P waves in lead V_1, negative P waves in lead aVL, and isoelectric P waves in lead I. There are few data regarding the P-wave morphology during CS arrhythmias; however, these results are compatible with arrhythmias originating from the left atrium (17).

Safety and effects of radiofrequency application in the CS. Radiofrequency energy delivery within the CS or its branches has been associated with collateral damage to the coronary arterial system (18), perforation (19), and CS stenosis. Arterial injury usually occurs to the branches of the right coronary or circumflex artery and is caused by the proximity of the arterial system to the coronary venous system. Perforation and tamponade probably occur as a result of endothelial disruption related to radiofrequency energy application. Despite increasing the temperature to 55°C and power to 70 W in some cases, no vascular injury or perforation occurred in patients in the current study. Although it is recommended that ablation for accessory pathways utilizing the CS be performed using low power (5 to 30 W) (11), one-half of the patients in the current study required ≥45 W for tachycardia termination. The fact that relatively high temperature and power settings were required does not necessarily imply that the tachycardia substrate was located at a more distal epicardial site. It is more likely that energy titration was required because of inadequate contact of the ablation catheter against the wall of the CS.

The absence of arterial injury may in part be related to the presence of adipose tissue between the CS and the circumflex artery (20). Furthermore, blood flow within the coronary arteries may serve as a heat sink that protects the arterial walls.

Because safety during the catheter ablation procedure is of the utmost concern, alternatives to radiofrequency energy for ablation of CS tachycardias may be considered. Although the experience is small, cryoablation has been used safely in ablation of accessory pathways using the CS musculature and in animal models (21–23). Another potential concern is that of injury to the esophagus (7) because it may be in close proximity to the course of the CS in some patients. The fat pad that invests the CS may insulate the esophagus from thermal injury during radiofrequency application in the coronary sinus.

Prior studies. A few case reports have documented the CS as the site of origin of focal (2,3) and macro–re-entrant tachycardias (4), and as a trigger for AF (24). A prior study (25) reported that in 96% of 25 patients who underwent an intraoperative radiofrequency ablation procedure for AF, atypical flutter developed postoperatively, and that the critical isthmus in 5 of the patients who underwent an electrophysiology procedure was in the CS.

A recent randomized study also reported that the mitral isthmus was a frequent target for atypical flutter after left atrial ablation (26). However, tachycardia termination did not require radiofrequency application in the CS in any of the patients. In contrast, CS ablation was required in approximately 25% of the patients in the present study. The reason for this difference is unknown.

Study limitations. A limitation of this study is that the prevalence of CS tachycardias after left atrial ablation for AF may have been underestimated. This is because during our initial experience with left atrial ablation for AF, not every atypical flutter that was observed during the procedure was mapped.

Coronary sinus venography was not routinely performed before ablation. Therefore, we cannot specify the sites of ablation within the CS relative to the vein of Marshall (27). Coronary angiography was not performed after the ablation procedures. Therefore, we cannot rule out asymptomatic coronary artery stenosis that may have occurred as a result of ablation in the CS. However, in a recent study coronary angiography was performed in a subset of patients who had undergone radiofrequency ablation in the distal CS with an irrigated-tip catheter, and no coronary artery stenoses were observed (28). Although there are no studies comparing ablation with irrigated-tip and 8-mm catheters in the CS, the former is likely to be safer and more efficacious in areas of low blood flow and high impedance.

Clinical implications. The main clinical implication of this study is that CS should be the first place to perform entrainment mapping in patients with atypical flutter after left atrial ablation for AF. This is true even when the ECG does not show inverted flutter waves in inferior leads. If a perfect PPI is found in the coronary sinus, it may avoid the need for transseptal catheterization and left atrial mapping or ablation. Catheter ablation in the CS appears to be safe and effective as long as the power is gradually titrated and the ablation catheter is withdrawn during energy delivery. Further studies are needed to assess alternative energy sources for ablation and the anatomic relationship between the CS and adjacent structures in hopes of minimizing possible complications.

Reprint requests and correspondence: Dr. Aman Chugh, Cardiology, TC B1 D140, 1500 East Medical Center Drive, Ann Arbor, Michigan 48109-0311. E-mail: achugh@umich.edu.

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